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ARTICLE

Correlation Between Vitamin D, Alkaline Phosphatase, and Lipid Profile in Type 2 Diabetes

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ABSTRACT

Background: Vitamin D is essential for immune system control, glucose metabolism, and musculoskeletal development. Conflicting findings have implicated vitamin D in dyslipidemia, and its deficiency has been associated with several clinical diseases.
Objective: This research sought to explore the connection between vitamin D level, alkaline phosphatase, and lipid profile in individuals with type 2 diabetes in contrast to those without diabetes.
Methods: The study was conducted as a case-control study in Mosul City/Iraq, from October 2024 to January 2025. Involved a total of 160 participants within the age range of 35 to 60 years old. The participants were divided into two categories: one group consisted of type 2 diabetics (referred to as cases, including 80 individuals), while the other group consisted of individuals (referred to as controls consisting of 80 individuals. The subjects' lipid profiles, glycemic control (hemoglobin A1c, fasting plasma glucose), alkaline phosphatase activity, and vitamin D levels were examined.
Results: The diabetes mellitus group (cases) showed significantly lower vitamin D (6.86 ± 2.3 ng/mL vs. 32.3 ± 12.14 ng/mL) and HDL-C levels (26.66± 9.85 mg/dL vs. 48.56± 9.66 mg/dL) and higher alkaline phosphatase activity(151.91± 22.47U/L vs. 61.18± 12.69 U/L), total cholesterol (213.43± 30.47 mg/dL vs.155.52± 13.7 mg/dL), LDL(137.2±, 31.06mg/dL, vs.81.85± 16.46mg/dL), VLDL (59.57±10.85mg/dL vs. 25.1±5.34mg/dL). Triglycerides (247.86± 54.27 mg/dL vs. 125.53± 26.73 mg/dL), and atherogenic index (10.24± 3.32 vs.5.33 ± 2.37) compared to the control. A positive relation was found between alkaline phosphatase and HbA1c ($r = 0.860$, $p < 0.001$), alkaline phosphatase and fasting plasma glucose ($r = 0.787$, $p < 0.001$) among cases. Lipid profile components, including total cholesterol correlated positively with HbA1c ($r = 0.876$, $p < 0.001$) and fasting plasma glucose ($r =0.836$, $p < 0.001$), LDL correlated positively with HbA1c ($r =,0.742$ $p < 0.001$) and fasting plasma glucose ($r = 0.752$ $p < 0.001$) while vitamin D levels were negatively related to total cholesterol($r =-0.609$, $p < 0.001$), LDL($r =-0.497$, $p < 0.001$), VLDL($r =-0.333$, $p =0.00251$), and triglycerides($r =-0.333$, $p =0.00251$).
Conclusion: Diabetic patients showed lower vitamin D levels, remarkably higher lipid profiles, and higher alkaline phosphatase compared to the control group. Strong correlations were observed between alkaline phosphatase, HbA1c, and fasting plasma glucose, indicating a potential link to glycemic control. Additionally, vitamin D levels showed negative correlations with total cholesterol, LDL, VLDL, and triglycerides in diabetic patients.

Introduction

Vitamin D (Vit-D) plays a crucial role in maintaining overall health by supporting bone integrity and enhancing immune system functions [1]. It contributes to the regulation of calcium and phosphorus levels, which are essential for skeletal development and strength [2]. Beyond bone health, Vitamin D is involved in numerous physiological mechanisms, including

modulation of immune responses, control of inflammatory processes, and cardiovascular protection. Emerging evidence has highlighted its influence on lipid metabolism and glycemic control [3], making it a subject of growing interest in type 2 diabetes mellitus (T2DM) research. T2DM is a metabolic condition marked by insulin resistance and chronic hyperglycemia [4]. According to Lima et al., patients with diabetes often exhibit reduced high-density lipoprotein (HDL) levels, accompanied by elevated triglycerides and total cholesterol concentrations [5].

The presence of dyslipidemia in diabetic patients increases their risk for cardiovascular complications [6]. Multiple investigations have shown a high incidence of vitamin D deficiency among individuals with T2DM, potentially disrupting lipid metabolism [7] [8] [9] [10]. Vit-D appears to influence enzymes related to cholesterol synthesis and the regulation of lipid equilibrium [11].

Additionally, vitamin D receptors (VDRs), found in various tissues such as pancreatic β-cells and fat cells, are believed to modulate vital metabolic activities, including insulin release and glucose absorption. Several reports suggest that a deficiency in vitamin D among T2DM patients may be linked to disturbances in glucose and lipid regulation [12] [13].

Given the concurrent prevalence of vitamin D deficiency and abnormal lipid profiles in individuals with T2DM, investigating the relationship between these variables in a localized population, such as patients from Mosul, Iraq, may provide region-specific insights and contribute to improved management strategies.

This study aims to investigate the association between serum vitamin D levels and lipid profile components in individuals with T2DM, compared to non-diabetic controls, to assess potential links and their relevance to diabetes management.

Methods

A case-control study was conducted in Mosul City, Iraq, between October 2024 and January 2025 utilizing random sampling. The study was approved by the Ethics Committee of the College of Medicine, University of Mosul. The study was conducted at Al-Wafa Specialized Center and Ibn Sina Teaching Hospital. Before participation, the participants were fully informed about the study's objectives and methodologies, and their informed consent was obtained.

The study included 160 participants aged 35 to 60 years, who were divided into two groups:

Group 1 (Cases): consisted of 80 individuals diagnosed with T2DM who were on hypoglycemic treatment.

Inclusion criteria comprised individuals aged 35 to 60 years with a confirmed diagnosis of type 2 diabetes mellitus (T2DM), irrespective of disease duration for T2DM cases were defined based on Diabetes Association (ADA) criteria Glycated Hemoglobin (HbA1c) $\geq$ 6.5%, according to the American Diabetes Association (ADA) guidelines, fasting blood glucose (FBG) > 126 mg/dL on two separate occasions [14]. Current use of diabetes medication (such as Metformin, sulfonylureas, or Insulin) was documented in the patient's medical records. Additionally, a clinical diagnosis of T2DM by an endocrinologist and a documented disease duration of at least six months before study enrollment were required.

Type 1 diabetes mellitus, individuals, with long term kidney or liver conditions, thyroid or parathyroid disorders and malignant diseases can lead to health issues and complications, present use of Vit-D supplements, taking medications that impact how the body processes Vit-D like corticosteroids or hormonal therapies that alter bone structure, presence of diabetes related complications such as heart or blood vessel issues that might affect bone strength. Pregnancy, since metabolic changes can impact the composition of bones.

Group 2 (control): Comprised of 80 healthy participants (healthy controls were defined based on the following criteria: Absence of diabetes mellitus, confirmed by fasting blood glucose < 126 mg/dL, according to recognized diagnostic criteria, absence of chronic diseases such as hypertension, liver disorders, kidney disease, or thyroid abnormalities, absence of clinical symptoms suggestive of any disease.

Participants in the control group had no history of diabetes and were matched to the case group by age and sex. They had no known hepatic, renal, thyroid, or parathyroid disorders and were not taking any vitamin D supplements. Control subjects were recruited from the hospital's outpatient department and included healthy relatives or companions of diabetic patients attending the facility during the study period. Only individuals who met all health-related eligibility criteria were included in the control group.

Data were collected through face-to-face interviews with the participants, followed by sample collection. Anthropometric measurements were also obtained, including calculations of body mass index (BMI) defined as weight in kilograms divided by the square height in meters [15].

Each participant underwent an interview, was advised to fast for 12-14 hours before providing a blood sample collection. Subsequently 7 millilitres of blood were extracted from every participant and separated into three separate tubes; Analysis of serum, for Vit- D Levels and Lipid Profile**:** After collecting four millilitres of blood in a serum separating gel tube and permit it to clot for 30 minutes at room temperature; the sample was then centrifugal at 3500 rpm for 12 minutes to obtain serum which was later divided into an Eppendorf tube and stored at -20°C until analysis for Vit-D levels and lipid profile parameters [16].

Serum lipid levels are measured, including total cholesterol, LDL, HDL, and triglycerides, using various methods. Total cholesterol is measured and calculated using a cholesterol oxidase enzymic method [17], while LDL is calculated using the Friedewald formula [18]. HDL (Good Cholesterol) is measured using precipitation/enzymatic assay [19]. Triglycerides are measured enzymatically by hydrolysing triglycerides into glycerol and free fatty acids [17]. VLDL (Very Low-Density Lipoprotein) levels are estimated using a specific formula (Triglyceride/5). The atherogenic index is calculated by dividing the total cholesterol level by the HDL level. A ratio below 3.5 indicates a lower risk of heart disease, while a ratio between 3.5 and 5 indicates an increased risk.

For the purpose of measuring HbA1c, two millilitres of blood were placed in an ethylene diamine tetraacetic acid (EDTA) test tube and gently shaken to guarantee proper mixing with the anticoagulant material, then measured by using the Turbidimetric Inhibition Immunoassay [20]. One ml was put into a sodium fluoride/potassium oxalate tube to obtain plasma for plasma glucose assessment. The determination of serum alkaline phosphatase (ALP) levels is based on a kinetic colorimetric method [21].

Statistical Analysis

All data were analyzed using SPSS version 22. For the parameters under investigation, an independent sample t-test was used to compare the means between the case and control groups (significant at $p<0.05$). Use Pearson's correlation to examine the connection between vitamin D and lipid profile.

Results

The majority of participants in the diabetic group were between 45-60 years (65%), and in the non-diabetic group was 53.25%. A smaller proportion was between 35-45 years, as 33.75% in the diabetic group and 46.75% in the non-diabetic group (Table 1). This distribution suggests that middle-aged individuals constitute the largest percentage in the diabetic group,

reflecting the higher prevalence of T2DM in this age category. In Group 1, 42.5% were males and 57.5% were females, while in Group 2, 52.5% were males and 47.5% were females (Table 1). The majority of the patients were above 5 years duration as 46 cases (57.5%), whereas under 5 years were 34 cases (42.5%) (Table .1). In the diabetic group, 31.25% of individuals had BMI 18.5-24.9 (normal weight), 46.25% had BMI 25-29.9 (overweight) and 22.5% had BMI ≥30(obese). In contrast, the majority of non-diabetic individuals 45% had BMI 18.5-24.9 kg/m2, 51.25% had BMI 25-29.9 kg/m2and only 3.75% had BMI ≥30 kg/m2 were overweight or obese (Table 1)

Table 1: Demographics of the studied groups

CHARACTERISTICS		GROUP1 (CASE) N=80		GROUP 2 (CONTROL) N=80	
		No.	**%**	**No.**	**%**
Age (years)	35-45	27	33.75%	38	46.75%
	45-60	53	66.25%	42	53.25%
Sex	Male	34	42.5%	42	52.5%
	Female	46	57.5%	38	47.5%
Duration of the disease	Under 5 years	34	42.5%	0	0%
	Above 5 years	46	57.5%	0	0%
BMI (kg/m^2)	18.5-24.9	25	31.25%	36	45%
	25-29.9	37	46.25%	41	51.25%
	≥30	18	22.5%	3	3.75%

The data in Table (2) presents the mean ± standard deviation (SD) values of the studied parameters in the case and control groups. A significant difference ($p < 0.001$) was observed between the two groups for all parameters.

Vit- D level in the T2DM group (6.86± 2.3) was significantly lower than that in the control group (32.3 ± 12.14), and ALP in the T2DM group (151.91± 22.47) showed a significantly higher activity compared to the control (61.18± 12.69). Total cholesterol in the T2DM group (213.43± 30.47) showed a significantly higher level compared to the control (155.52± 13.7). HDL-C shows a significant reduction in the T2DM group (26.66±9.85). Higher LDL (137.2±31.06), VLDL (59.57±10.85), triglyceride (247.86± 54.27), and atherogenic index (10.24± 3.32) in the T2DM group.

Table 2: The values of Vit D, ALP, and lipid profile in the case (T2DM) and control.

Parameters	**Cases (N= 80)**	**Controls (N= 80)**	**p- value**
Vit- D (ng/mL)	6.86± 2.3	32.3 ± 12.14	<0.001
ALP (U/L)	151.91± 22.47	61.18± 12.69	<0.001
Total cholesterol (mg/dL)	213.43± 30.47	155.52± 13.7	<0.001
HDL-C (mg/dL)	26.66± 9.85	48.56± 9.66	<0.001

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LDL-C (mg/dL)	137.2± 31.06	81.85± 16.46	<0.001
VLDL-C (mg/dL)	59.57±10.85	25.1± 5.34	<0.001
Triglycerides (mg/dL)	247.86± 54.27	125.53± 26.73	<0.001
Atherogenic index	10.24± 3.32	5.33 ± 2.37	<0.001

The t-test is significant at $p \leq 0.05$.

The correlation between Alkaline Phosphatase activity, HbA1c, and Fasting Plasma Glucose

Table 3, Figure 1, presents the positive correlation between ALP levels with HbA1c ($r = 0.860$, $p < 0.001$), and between ALP levels with FPG ($r = 0.787$, $p < 0.001$) among the studied groups. While the correlation in the control is not significant between ALP levels with HbA1c (r=0.795, p=0.0134), and ALP levels with FPG (r=622, p=0.0729).

Table 3: The correlation between Alkaline Phosphatase, HbA1c, and Fasting Plasma **Glucose.**

	T2DM Group1 n=80		**Control Group1 n=80**	
	R	*p*-value	r	*p*-value
HbA1c	0.860	<0.001	0.795	0.0134
FPG	0.787	<0.001	0.622	0.0729

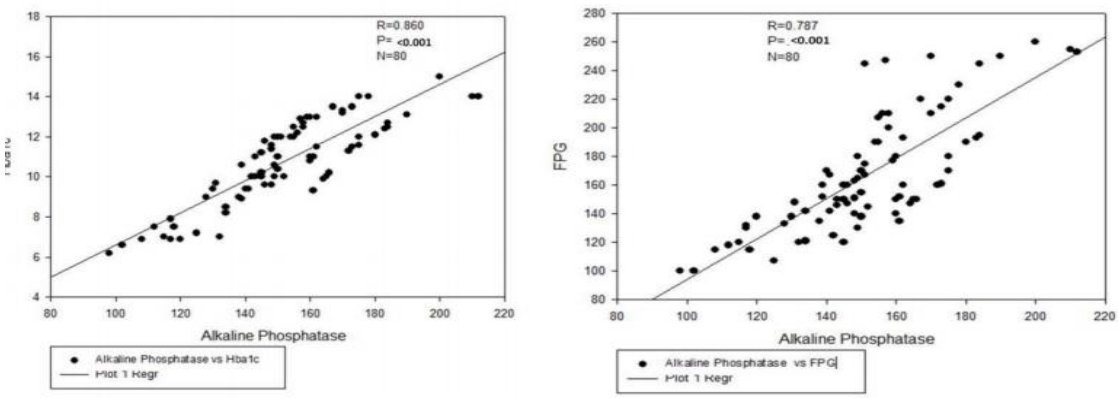

Figure 1: Correlation of the Alkaline Phosphatase level with HbA1c and FPG in the Case group.

The correlation of the lipid profile with HbA1c and Fasting Plasma Glucose:

Table 4 presents the correlation between the lipid profile, HbA1c, and FPG in the T2DM group: Total cholesterol showed a direct correlation with HbA1c ($r = 0.876$, $p<0.001$) and FPG ($r= 0.836$, $p<0.001$), LDL-C exhibited a positive correlation with HbA1c ($r= 0.742$, $p= <0.001$) and FPG ($r= 0.752$, $p=<0.001$).

VLDL-C and triglycerides had similar positive correlation patterns with HbA1c ($r= 0.368$, $p=<0.001$) and FPG ($r= 0.264$, $p<0.001$). HDL-C displayed a non-significant negative correlation with HbA1c ($r = -0.0398$, p 0.726) and FPG ($r = -0.0884$, $p = 0.436$). Atherogenic index shows a significant positive relationship with HbA1c ($r = 0.264$, $p = 0.0179$) and Atherogenic index with FPG ($r = 0.290$, $p = 0.00904$) in the T2DM group.

Table 4: Correlation of the lipid profile with HbA1c and Fasting Plasma Glucose FPG among diabetic patients.

T2DM (N=80)	Hba1c		Fasting Plasma Glucose FPG	
	r	P value	R	P value
Total cholesterol	0.876	<0.001	0.836	<0.001
LDL-C	0.742	<0.001	0.752	<0.001
VLDL-C	0.368	<0.001	0.264	0.0181
HDL-C	-0.0398	0.726	-0.0884	0.436
Triglycerides	0.368	<0.001	0.264	0.018
Atherogenic index	0.264	0.0179	0.290	0.00904

There was a significant negative connection between Vitamin D and total cholesterol, LDL.
C, VLDL-C, and triglycerides as (r=-0.609, -0.497, and -0.333) respectively, but there was a non-significant positive connection between Vit-D and HDL-C (r=0.0569, p=0.616) (Table 5).

Table 5: Correlation of the serum Vit-D level with lipid profile among diabetic patients**.**

	T2DM (N=80)	
	r	*p*- value
Total cholesterol	-0.609	<0.001
LDL-c	-0.497	<0.001
VLDL-c	-0.333	0.00251
HDL-c	0.0569	0.616
Triglycerides	-0.333	0.00251

Discussion

Type 2 diabetes mellitus (T2DM) is a prevalent chronic metabolic disorder that involves insulin resistance, impaired insulin secretion, and persistent hyperglycemia. It is frequently accompanied by abnormalities in lipid metabolism, known as diabetic dyslipidemia, which increases the risk of cardiovascular complications [22] [23]. In recent years, vitamin D has emerged as a potential modulator of glucose and lipid homeostasis. Vitamin D receptors (VDRs) are located in key metabolic tissues such as pancreatic β-cells and adipose tissue, where they may affect insulin production, secretion, and sensitivity [24]. Numerous investigations have indicated that individuals diagnosed with type 2 diabetes mellitus (T2DM) frequently exhibit insufficient vitamin D levels. This insufficiency may be linked to compromised metabolic regulation, as it is associated with disturbances in lipid metabolism and poor glycemic control [25] [26]. Accordingly, the current study seeks to evaluate the relationship between serum vitamin D concentrations, lipid profile components, and indicators of glycemic regulation in patients with T2DM in Mosul, Iraq, aiming to generate insights specific to this regional population.

According to findings by Tarfeen et al., vitamin D plays a significant role in promoting insulin synthesis by regulating calcium levels within cells—a process essential for the release of insulin granules [27]. Moreover, vitamin D may enhance insulin sensitivity by influencing the expression of insulin receptors in insulin-responsive tissues such as skeletal muscle and adipose tissue [28] [29] [30]. A deficiency in vitamin D among diabetic individuals has been shown to impair insulin secretion and β-cell function, primarily due to inadequate calcium entry into the cells, which is crucial for the release of insulin-containing vesicles [31]. Additionally,

insufficient vitamin D is linked to decreased insulin receptor expression and weakened signalling pathways, thereby contributing to insulin resistance. This situation is exacerbated by persistent inflammation and oxidative stress, both of which are frequently observed in diabetes [32].

In the present study, total cholesterol levels were found to be significantly elevated among diabetic participants compared to the non-diabetic control group. This elevation reflects an increased likelihood of developing dyslipidemia-related complications, including atherosclerosis, coronary artery disease (CAD), and other cardiovascular disorders. One of the proposed mechanisms underlying this association involves the upregulation of hepatic cholesterol synthesis in insulin-resistant individuals. Specifically, insulin resistance can enhance the activity of hydroxymethylglutaryl coenzyme A (HMG-CoA) reductase—the key enzyme controlling cholesterol biosynthesis—leading to elevated cholesterol production and diminished clearance [33]. Impaired insulin signalling is associated with reduced expression of LDL receptors, leading to decreased hepatic uptake and clearance of circulating cholesterol, thereby promoting hypercholesterolemia [34]. Oxidative stress and inflammation induced by chronic hyperglycemia contribute to endothelial dysfunction and the oxidation of LDL particles, making them more atherogenic [35]. HDL-C is known for its cardioprotective role through reverse cholesterol transport, where it facilitates the removal of cholesterol from peripheral tissues and transports it back to the liver for excretion. Even if HDL-C levels remain relatively stable, diabetes is often associated with dysfunctional HDL-C, characterized by reduced antioxidant and anti-inflammatory properties. Diabetic HDL-C tends to lose its protective properties and may become pro-inflammatory, contributing to endothelial dysfunction [36].

Increased glycation and oxidation of HDL-C in hyperglycemic conditions can impair its cholesterol efflux capacity, making it less effective at preventing atherosclerosis [37]. Lower ApoA-I content: Apolipoprotein A-I (ApoA-I), the primary protein component of HDL-C, is often reduced in diabetics, further compromising HDL-C functionality [38]. Diabetic dyslipidemia is often characterized by small, dense LDL (sdLDL) particles, which are more atherogenic than larger, more buoyant LDL particles due to several reasons, including increased susceptibility to oxidation, leading to the formation of oxidized LDL (ox LDL), a key player in atherosclerosis [39]; reduced LDL receptor affinity, as these particles exhibit decreased binding to LDL receptors, leading to prolonged circulation and increased arterial deposition [40]; and especially these particles more easily penetrate the endothelial layer of blood vessels, where they contribute to foam cell formation and plaque development [41].

Even if the total LDL-C concentration remains similar between diabetics and non-diabetics, the predominance of sd LDL in T2DM makes LDL more atherogenic. A significantly higher VLDL-C concentration was observed in diabetics, along with elevated triglyceride levels. These findings suggest profound alterations in lipid handling and increased risk of metabolic complications. Mechanisms of hypertriglyceridemia in T2DM are that insulin resistance conduct to increased hepatic production of triglycerides and VLDL, contributing to hypertriglyceridemia. Reduced Lipoprotein Lipase (LPL) Activity, an enzyme responsible for hydrolyzing triglycerides in chylomicrons and VLDL, is often impaired in diabetes, leading to delayed clearance of triglyceride-rich lipoproteins in adipose tissue [42]. However, in insulin-resistant states, uncontrolled lipolysis results in excess free fatty acids (FFA) being released into circulation, which are subsequently taken up by the liver and converted into triglycerides [43]. Formation of atherogenic remnants, as elevated triglycerides promote the exchange of triglycerides for cholesterol esters in LDL and HDL via cholesterol ester transfer protein (CETP). This results in triglyceride-enriched LDL and HDL, which are prone to hydrolysis, leading to small dense LDL and dysfunctional HDL particles [44].

The atherogenic index was higher in diabetic patients compared to controls. This indicates a higher risk of atherosclerosis and cardiovascular events in the diabetic group. The strong association between vitamin D deficiency, dyslipidemia, and an elevated atherogenic index supports the hypothesis that vitamin D may have a protective role against metabolic and cardiovascular complications in diabetes [45].

The present study demonstrates a high positive correlation between serum ALP and both HbA1c and FPG in patients with T2DM. The present study demonstrated a strong positive association between alkaline phosphatase (ALP) activity and HbA1c levels, indicating that increased ALP may serve as an indicator of poor glycemic regulation in individuals with type 2 diabetes mellitus (T2DM). This observation aligns with earlier research that identified elevated ALP as a potential marker of underlying subclinical inflammation, oxidative stress, and endothelial dysfunction—factors commonly observed in diabetic patients and known to disrupt metabolic balance [46] [47].

The rise in ALP levels among T2DM patients may be partly attributed to systemic inflammatory responses and their hepatic origin, especially in light of the high prevalence of non-alcoholic fatty liver disease (NAFLD) within this population [48]. Additionally, increased ALP levels have been associated with insulin resistance and elevated cardiovascular risk, suggesting its potential utility as a prognostic marker for disease complications and progression [49].

In this study, significant correlations were also observed between lipid profile parameters and glycemic markers such as HbA1c and fasting plasma glucose (FPG). These findings support the notion that elevated cholesterol levels, particularly hypercholesterolemia, may reflect long-term poor glycemic control. Low-density lipoprotein cholesterol (LDL-C) showed a particularly notable positive correlation with both HbA1c and FPG, reinforcing prior research that highlights LDL-C as a key factor in the pathogenesis of atherosclerotic changes in patients with T2DM [50]. Likewise, VLDL-C and triglycerides were significantly correlated with both HbA1c and FPG, though the correlations were moderate, supporting evidence that elevated triglycerides and VLDL-C are common in insulin-resistant states and are associated with increased cardiovascular risk [51]. Conversely, HDL-C was negatively but not significantly correlated with HbA1c and FPG, which is consistent with previous findings that low HDL-C is a hallmark of diabetic dyslipidemia, but its levels may not always correlate linearly with glycemic indices [52].

There was a positive and significant correlation between HbA1c and FPG and the atherogenic index, a derived ratio that represents the equilibrium between atherogenic and protective lipids. This reinforces its potential utility as a predictive marker for cardiovascular complications in diabetic patients, particularly when used in conjunction with traditional lipid parameters [53]. The present study reveals a significant negative correlation between Vitamin D levels and key lipid profile components among patients with T2DM. These findings are in agreement with prior research suggesting that Vit-D may exert lipid-lowering effects, potentially through its role in improving insulin sensitivity and modulating inflammatory processes [54] [55]. Vit-D has been shown to influence lipid metabolism by affecting the expression of genes involved in cholesterol synthesis and clearance, as well as by reducing hepatic triglyceride production [56].

Furthermore, hypovitaminosis D is frequently observed in people with T2DM and is often associated with metabolic disturbances, including dyslipidemia and obesity, which may exacerbate cardiovascular risk [57]. Interestingly, while HDL-C showed a positive correlation with Vit-D, this association was not statistically significant. This aligns with some studies indicating that although Vit-D supplementation may improve HDL levels modestly, the effect is often variable and influenced by factors such as baseline Vit-D status, BMI, and genetic predispositions [58].

Conclusion

Patients with type 2 diabetes mellitus demonstrated significantly lower serum vitamin D levels and higher concentrations of lipid profile components—including total cholesterol, LDL-C, VLDL-C, and triglycerides—compared to healthy controls. Moreover, vitamin D levels exhibited negative correlations with these lipid parameters in the diabetic group. These findings suggest a potential role of vitamin D in lipid metabolism and its relevance to the metabolic profile of individuals with diabetes. Routine assessment of vitamin D status may be beneficial, particularly in patients with poor glycemic control, and may support preventive strategies aimed at reducing the risk of metabolic complications through vitamin D supplementation and adequate sun exposure.

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Conflicted interest

Regarding this manuscript's publication, the authors declare that they have no conflicts of interest.

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